COMPARISON OF EFFECTS INDUCED BY FORMALDEHYDE EXPOSURE ON ALVEOLAR AND BRONCHIAL EPITHELIAL CELLS IN VITRO

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Background and Aims Exposure to formaldehyde (FA) at environmental concentrations is suspected to be associated to asthma and respiratory disorders. Nevertheless, the physiopathology of this relation is still unclear and experimental data are needed. In vitro experimentation is complementary to epidemiological studies. However experimental procedure parameters need to be adapted, such as the choice of target cells of respiratory tract. The aim of this study was to compare the inflammatory response of FA exposure at environmental level on alveolar and bronchial epithelial cells.

Methods
Human alveolar (A549) and bronchial (BEAS-2B) epithelial cells were exposed at the air-liquid interface in a Vitrocell exposure module to FA (50 µg/m3) (or air for control) during 30 min. After 24h post-incubation, cellular viability was assessed using LDH release. Two chemokines (IL-8, MCP-1) were assayed in the apical supernatant by ELISA. In addition, to modulate inflammatory response, pre-sensitization was performed using LPS (5 µg/mL) (to mimic a bacterian activation), TNFα (1 ng/mL) or macrophages conditioned media (to mimic the macrophages/epithelial cells cooperation) during 16h before exposure.

Results
Whatever the cell type, cell viability was unaffected by air or FA exposure compared to cells not exposed at the air-liquid interface. Without presensitization or after LPS presensitization, FA had not effect on A549 or BEAS-2B, compared to air-exposed cells. FA exposure, after TNFα presensitization, resulted in an inflammatory response, an increase of IL-8 production by A549 cells; after macrophage conditioned media presensitization, an increase of IL-8 production by A549 cells, and decrease of MCP-1 production by BEAS-2B cells was observed.

Conclusions
After presensitization FA exposure at environmental levels provokes an inflammatory response in epithelial cells of the respiratory tract. Moreover, this response is differently modulated depending on the level of the cells in the respiratory tract.